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AUTHOR Roberts, David Harrill
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ABSTRACT

For many years dyslexia has been incorrectly applied to those who have demonstrated difficulty in learning to read. Given the proper guidance and opportunities for becoming sensitive to demonstrations of the workings of language and engaging their minds in learning, many students will overcome their so-called learning disabilities. However, there are some language disorders that have a neurophysiological basis. One study suggests a lack of cerebral asymmetry may lead to dyslexia. Computerized axial tomography (CAT scan) has given support to the hypotheses that to the extent that learning to read involves gestalt perception and right hemispheric processing, abnormal specialization of the right hemisphere may also be an instrumental factor in developmental dyslexia. Other researchers have combined psycholinguistic methods with neurolinguistic methods to suggest that learning to use a graphic code comes only after knowing the correspondences between acoustics and the graphic code, the grapheme-phoneme correspondence rules. When these rules break down because of a neural coding problem, alexia and agraphia may occur. Other studies show that word-blindness (alexia without agraphia) may be caused by a variety of brain insults. A knowledge of the brain functions of language acquisition and development in out-of-school life will lead to a development of improved language arts pedagogies. (HOD)

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# Neurological Processes and Reading Pathology: Knowing About Children and Reading Dysfunction

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David Harrill Roberts

Assistant Professor of English

Bluefield State College

Bluefield, WV 24701

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Introduction

'Dyslexia' is a term that has been misused for many years and incorrectly applied to students and others who have demonstrated any difficulty at all in learning to read, even when no physical pathology is present. The purpose of this paper is to expose the tyranny of the overuse and misuse of the term as applied in our schools from kindergarten through college. Students and teachers alike all to often fall back on 'dyslexia' as the reason for failure of students to read well, but Frank Smith (1978) does "not believe that failure should be attributed to dyslexia, a disease," Smith adds, "that only strikes children who cannot read and which is invariably cured when they can read" (Smith 1978, p. 193). Children are born with inquisitive minds, products of brains that are biologically suited for learning, a task the brain does very well. Telling children that they have a learning disability creates a learning disability (Smith 1981), so educators must use extreme caution in talking about supposed learning disabilities lest they create them.

This paper is an attempt to inform English teachers of the various classifications of language disorders arising from neuropathologies to head off, by education, any premature diagnosis of dyslexic forms in students who read less well than we would wish. We begin with a highly truncated introduction to the language areas of the

human brain, then discuss two types of alexia, developmental dyslexia and word-form dyslexia. Finally, we end with a call for the interdisciplinary training of educators and researchers who will advance pedagogies based on solid scientific findings rather than hunches, guesses, and gut feelings.

Brain Anatomy and Human Language

The human brain is divided into three main parts, the forebrain, the midbrain, and the hindbrain. The forebrain is composed of the cerebral cortex, basal ganglia, corpus callosum, and the limbic system. Divided into two parts known as the right and left cerebral hemispheres, the cerebral cortex is the most studied part of the living brain because of its relative accessability and the many controls over human activity it demonstrates. The left cerebral cortex is normally dominant for language activities and the right for other kinds of activity, such as facial recognition and spatial relationships. 'Hemispheric dominance' and 'lateralization' are terms commonly associated with theories and facts related to this division of activities. The midbrain and the hindbrain developed earlier in the evolutionary process and are only peripherally interesting to studies of language and cognition. The three parts of the mature brain combined, about 3½ pounds in weight, contain as many as one trillion neurons, each with as many as ten thousand connections called synapses. At each synapse the electro-chemical transfer of information is made via thirty different kinds of transmitter molecules in any combination for a theoretical total of ten quadrillion connections, each with

the potential for many millions of message units according to the quantity and type of transmitter molecule in combination with intensity of electrical spike, yielding, theoretically at least, more than $2.0589 \times 10^{60}$ bits of information in the 'fully programmed' human brain. It is no wonder that the language competence of humans can generate sentences to express an infinite number of thoughts in an infinite variety of ways.

The central language system is located in the cerebral cortex of the left hemisphere and is made up of areas responsible for certain production and comprehension tasks. Broca's area, located on the third gyrus of the left frontal lobe, is generally acknowledged as the primary area for language production of speech. Wernicke's area, located on the posterior temporal lobe, is thought to be the auditory decoding region. The angular gyrus, located adjacent and posterior to Wernicke's area, relays sensations from the primary visual cortex to Wernicke's area during reading. Broca's and Wernicke's areas are connected via the arcuate fasciculus, a bundle of millions of neurons. The two hemispheres are connected via the corpus callosum, especially important in transferring visual language data from the right hemisphere to the left, as we shall discuss later.

## Aphasia and the Competence-Performance Distinction

Language performance is a complicated system of related functions working rather independently, as demonstrated by the many different manifestations of aphasic disorders. Weigl and Bierwisch (1973)

have hypothesized that competence in some aphasics is undisturbed, that only performance is interferred with. They support their position with conclusions drawn after observation of aphasics and "deblocking" experiments employing a psycholinguistic method of performance-switching, stating that they consider aphasia a disturbance of the access to the knowledge of language because a) some, but not all, speech components are disturbed; b) performance fluctuates from day to day; c) sometimes aphasics can write but not speak, speak but not write, comprehend aurally but not visually, or read but not understand verbal language. Even so, some aphasic disturbances can affect some aspects of competence: "some aphasic phenomena can be classified and described in terms of the structure of competence, i.e., in terms of grammatical and lexical structure" (Weigl and Bierwisch 1973, p. 15).

Demon Dyslexia

Dyslexia has been blamed for the failure of many children to learn to read well, as Frank Smith (1978) has so clearly stated.

> There are no evident visual defects that are specific to reading, but this does not mean that there are no general visual anomalies that will interfere with learning to read. Children who need glasses will not find reading easy until their sight is corrected. The few children who have difficulty learning to understand speech, or learning anything, may also find learning to read difficult.
>
> But there is no convincing evidence that children who can see normally, with or without classes, and who have acquired a working competence in the language spoken around them, might be physically or congenitally incapable of learning to read . . . Children do not learn to read if they have the wrong idea of what reading is about, if they have learned - or been taught - that reading does not make sense (p. 193).

Still, others find neurological or psychological evidence for several varieties of dyslexia, a term broadly applied to a wide variety of reading deficits. Espir and Rose, British neurologists, define developmental, or specific dyslexia as a reading deficit of more than two years behind mental age, yet some educators doubt the existence of specific dyslexia because "the range of normality is so wide" (Espir and Rose 1976, p. 35). With developmental dyslexia, most subjects also have subnormal intelligence, a marked decrease in overall learning ability, tend to reverse and transpose letters at a later stage than normal, and make reading mistakes in an inconsistent manner. One or more of the following reading difficulties, say Espir and Rose, are manifested by so-called dyslexic patients:

1. Inability to work out the pronunciation of a strange word.
2. Failure to see likenesses and differences in forms of words.
3. Failure to see differences in shapes of letters.
4. Making reversals.
5. Failure to keep the place.
6. Failure to read from left to right.
7. Poor concentration.
8. Failure to read with sufficient understanding.

(Espir and Rose 1976, p. 36)

Frank Smith (1981), on the other hand, indicates that researchers in the 1980s should look not at the underlying brain mechanism for understanding failure to learn to read, but to look for instructional inadequacies instead of excusing classroom failure by appealing to

congenitally or environmentally related factors. The important question for Smith is not what underlying brain processes defeat learning attempts:

> The answer cannot be in the nature of the brain itself, for if it can learn one thing, why not everything? I cannot believe that there are brain cells or processes that specialize in spelling, punctuation, arithmetic, mechanics, physics, botany, or any of those things many otherwise unexceptional people learn while otherwise competent people fail. The answer must have something to do with the brain's approach to learning, rather than with any innate and specific inability to learn. It must be possible for the brain to learn in such a way that certain areas of learning in effect become closed off.
>
> (Smith 1981, p. 108)

People learn, Smith goes on, through demonstrations, engagement, and sensitivity. Demonstrations show how something is done. Engagement is the mind pondering, attending to a particular demonstration. In reading, the mind may be engaged in a demonstration of how a word is spelled or how a phrase has been well formed, or how a character, fictional or historic, conducts conversation at a cocktail party. Sensitivity is the expectancy of learning, which Smith says each brain naturally possesses. That expectancy, that anticipation of engaging the mind in a demonstration, can be effectively destroyed through experience, e.g., through a teacher telling a student that reading and writing are difficult to learn. Since many children are taught by peers, parents, and professional educators that learning to read is difficult, many fail to learn to read well; their sensitivity to learning is lost (Smith 1981 pp. 108-111). Not one to

leave things half said, Smith goes on to say that "early diagnosis" of language-learning difficulties may serve only to "transform a possibility into a probability" (Smith 1981, p. 112).

Empirical Evidence for Underlying Pathologies

In spite of Smith's warning about the dangers of diagnosing dyslexic forms, there is hard data confirming the existence of neurological reasons for reading difficulty - and early diagnosis may be a way to help the true dyslexic overcome his/her handicap. One study suggests a lack of cerebral asymmetry may lead to dyslexia. As reported by Gerald Leisman and Maureen Ashkenazi (1980), some subjects have been studied through noninvasive techniques such as computerized axial tomography (CAT scans). Leisman and Ashkenazi have clear evidence to support the hypothesis that "to the extent that learning to read involves gestalt perception and right hemispere processing, abnormal specialization of the right hemisphere may also be an instrumental factor in developmental dyslexia" (Leisman and Ashkenazi 1980, p. 157). Asking whether there is an anatomical substrate in dyslexia and whether there exists independence or coherence, electrophysiologically, between the left and right cerebral hemispheres of developmental dyslexics, Leisman and Ashkenazi studied 20 dyslexic children ranging in age from 7 to 10.9 years, 19 boys and 1 girl who were attending the Institute for Learning Development at the Eye Institute of New Jersey. The subjects had a mean age of 7.6 years, mean WISC-R I.Q. score of 104.23, mean Verbal I.Q. score of 98.28, and mean Performance I.Q. score of 104.41.

Each subject had good visual acuity (20/30 or better), no evidence of ocular pathology, no mental retardation, and no other signs of neurological pathology, yet each was retarded more than two years in reading age (Espir and Rose's criterion for developmental dyslexia). The control group of 16 boys and 4 girls had a mean age of 8.2 years, mean WISC-R I.Q. score of 103.9, mean Verbal I.Q. score of 101.69, and mean Performance I.Q. score of 98.47. Like the study group, the subjects had good vision, no ocular or neurologic pathologies, and no mental retardation. Each member of the control group read at grade level or better.

Leisman and Ashkenazi recorded EEG activity on all 40 subjects at eyes-closed resting state and eyes-opened resting state while undergoing continuous performance tests and while viewing projected items from the Stanford-Binet tests, recording naming latencies during the Stanford-Binet projections. They also recorded EEG activities while the subjects read grade-appropriate paragraphs from the Spache Diagnostic Reading Tests. The raw EEG data were recorded and stored for computer analysis. On computer analysis, the data showed that the dyslexics demonstrated greater electrical activity in the 3-7 Hz and 16-28 Hz bands, with peaks around 6, 7, 10, and 24 Hz. Normals showed greater electrical activity in the 9-12 Hz band and a strong peak in the alpha band around 10 Hz, unlike the dyslexics. All other data gathered showed no significant difference between the dyslexics and the normals; the only notable differences were in the parieto-occipital regions; the suggestion here is that the functional

organization of the parieto-occipital regions in dyslexics is different from normals. Further indication of differences in functional organization is the finding that in normals large interhemispheric coherences occured over the parieto-occipital regions, but in the dyslexics a great deal of ipsilateral coherence occured over the same regions.

To determine differences in the anatomical substrate of the functional independence of the hemispheres, two normals and eight dyslexics were submitted to CAT scans. Leisman and Ashkenazi expected the dyslexics to have abnormally-shaped parieto-occipital regions, an expectation confirmed by the CAT scans. The two normal subjects had wider left hemisphere parieto-occipital regions; two of the dyslexics had wider right hemisphere parieto-occipital regions and six had parieto-occipital regions of equal size. Leisman and Ashkenazi concluded from their study of the 20 normal and 20 dyslexic subjects that a "master-slave" relationship of the left-to-right hemispheres exists in respect to language in normal humans; that normals' hemispheres share a great deal of language information in reading, and the hemispheres of dyslexics do not; and that the right hemisphere in dyslexics is more autonomous that in normals with respect to visual language stimuli - perhaps even equal to the left.

Other research, that of Egon Weigl (1975) in particular, has combined psycholinguistic methods with neurolinguistic methods rather than to look strictly at evidence from either field. According to Weigl learning to use a graphic code, a graphic language mode, comes only

after knowing the correspondences between acoustics and the graphic code, the grapheme-phoneme correspondence rules. The grapheme-phoneme correspondence rules provide indirect relationships, "correspondences," between phonemes and clusters of letters known as graphic units. Each graphic unit is in a context-dependent relationship to a phonemic or phonetic unit, with the exception of graphemic ambiguities, e.g., raze-raise, beach-beech, altar-alter, sign-sine. When the grapheme-phoneme correspondence rules break down because of a neural coding problem caused by a lesion or other brain dysfunction, alexia and agraphia may occur, according to Weigl.

Another language deficit with clear documentation by a variety of researchers is pure word-blindness, or alexia without agraphia. Samuel H. Greenblatt (1973, 1976, 1980), a medical researcher in the Department of Neurosciences at the Medical College of Ohio at Toledo, has made extensive studies of alexics, agraphics, and hemianopsics, concluding in each case that alexias caused by a variety of brain insults can easily result in pure word-blindness. Elizabeth Warrington and Tim Shallice (1980), from the National Hospital in London, recently reported case studies of two patients with acquired dyslexia manifested in letter-by-letter reading in the absence of whole word reading, which was apparently impossible for the patients. Warrington and Shallice conclude that word-form dyslexia results from "damage to the system through which a visual word-form is attained" (p. 110). Warrington and Shallice came to this conclusion after a series of six experiments. The first three experiments were designed to determine the extent of possible damage to the visual

pathways that would result in a deficit in peripheral visual factors: angle of attention span, selective attention, and visual short-term memory. Warrington and Shallice found that visual and perceptual factors could not be blamed for the subjects' reading difficulties. In the second group of three experiments Warrington and Shallice conclusively proved that the patients were able tc read only letter-by-letter. The fourth experiment guaged reading speed and the effect of word length on reading speed. The finding was that reading speed in the two subjects examined was directly related to word length, indicating letter-by-letter reading. Script reading, extremely difficult in letter-by-letter strategies, would force the subjects to read whole words if they could. One subject read script much slower with more errors and the other read script so poorly that only her errors could be recorded; the number of errors was significantly higher when trying to read the script than when trying to read printed words. The sixth experiment, tachistoscopic reading, showed that both subjects read tachistoscoptically-presented words with very little success when high-frequency words of 3 and 6 letters were presented for 200 milliseconds. According to the research of Elizabeth Warrington and Tim Shallice (1980) pure word-form dyslexia does indeed exist.

Research and Academic Training Recommendations

At a symposium on the neurological bases of language disorders in children sponsored by the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the National Institute of Health (NIH) about three years ago, attempts were made to learn

whether the study of brain organization in normal adults would shed any light on the study of brain organization in language-impaired children. One of the results was that patterns of language performance in split-brain adults and those of acquired aphasia patients due to left hemisphere injury in childhood are very similar. Based on these and other findings, Christy Ludlow (1979) makes the following "recommendations for research on the neurological bases of language disorders in children":

1. The need for multidisciplinary collaboration calls for the cooperation of neuroscientists, neurolinguists and others to conduct neurophysiological and neuroanatomical studies. The suggestion for post-doctoral training of specialists is encouraged.

2. The need for longitudinal studies of individuals has been demonstrated as necessary in understanding the process of language development in language-impaired children.

3. The need for multidimensional descriptive data requires simultaneous measurement of psychological and neurological factors to allow fuller use of the information available.

4. Neurological studies of verbal processing should be applied to investigations of language-impaired children.

5. The need for anatomic studies, especially cytoarchitectonic studies of the central language system was demonstrated by Norman Geschwind.

Finally, Ludlow called for support of pre-doctoral research training, stressing that it "should be multidisciplinary in nature, providing candidates with exposure to various disciplines such as neuroscience, psycholinguistics, and experimental psychology. Programs should be across departments allowing candidates to take courses from specialists in each of the disciplines directly" (p. 190).

Echoing the sentiments of the participants of the symposium, I suggest that all teachers of English become aware of the vital research in the areas of normal and impaired language development.

Summary

In this paper I have tried to introduce a very complex subject to the teacher of English at all levels, kindergarten through graduate. The purpose has been to encourage educators to recognize the possibility of neurologically-impaired language development in children without falling into the trap of a premature diagnosis, thereby limiting the learning experiences of the student who may merely be learning at a rate slower than the rest of the class. Given the proper guidance and opportunities for becoming sensitive to demonstrations of the workings of language and engaging their minds in learning, as Frank Smith (1981) says, many students will overcome their so-called learning disabilities. We have reviewed some of the research into specific cases of developmental dyslexia, cases with definite neurological deficits, to demonstrate the existence of the neurophysiological origins of some language disorders. We have also reported on a recent national symposium that calls for the multidisciplinary study of language pathology for identification and proliferation of solutions to language-learning difficulties. The hard science approach is best when tempered by Frank Smith's (1981) pedagogical approach, "The human brain learns all the time. But in the process of learning particular things, or even before the learning of these things has begun, the brain may

learn that these things are not worth learning, or are unlikely to be learned. Everyone concerned with practice and research in education should perhaps develop more sensitivity to the nature of the demonstration with which children might become engaged at school" (Smith 1981, p. 112).

I submit that knowing children and how their brains develop, and research on deviant and normal language development demonstrate an increased sensitivity not only to pedagogies which are concerned with demonstration and engagement in the classroom, but also increase our knowledge of how engagement with language arts demonstrations out of school is accomplished by the brain. Knowing the brain functions of language acquisition and development in out-of-school life will help us to develop improved language arts pedagogies.

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